L Number	Hits	Search Text	DB	Time stamp
10	45594	chlorophenyl	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 15:24
17	0	9419321.pn. and chlorophenyl	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 15:24
24	41560	disulfide	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 15:24
31	0	9419321.pn. and disulfide	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 15:24
3	2	9419321.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 15:33
-	55	aldrithiol	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 15:18
-	16057	retrovir$4	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 10:32
-	17	aldrithiol and retrovir$4	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 10:33
-	1037	zinc adj finger$1	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 10:34
-	2	aldrithiol and (zinc adj finger$1)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 10:34
-	4	832236.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 10:56
-	62	karlstrom.in.	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 10:56
-	5223	hiv and protease	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 10:57
-	4	karlstrom.in. and (hiv and protease)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 10:59

-	2	9419321.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 15:23
-	7	"2127-03-9"	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 15:18
-	0	9419321.pn. and "2127-03-9"	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 15:19
-	2	9419321.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/09/24 15:19

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     FILE 'MEDLINE' ENTERED AT 09:28:14 ON 24 SEP 2001
L1          6164 S ZINC(W)FINGER
L2        103154 S HIV
L3           253 S L1 AND L2
L4         23679 S DISULFIDE?
L5            18 S L3 AND L4
L6          3043 S MALEIMIDE?
L7             0 S L3 AND L6
L8          1977 S HYDRAZID?
L9             0 S L3 AND L8
L10            0 S ALPH(W)HALOGENAT? KETONE?
L11            3 S HALOGENAT? KETONE?
L12            0 S L2 AND L11
L13            0 S 2127-03-9/CRN

     FILE 'REGISTRY' ENTERED AT 10:18:43 ON 24 SEP 2001
L14            1 S 2127-03-9/RN
                 SET NOTICE 1 DISPLAY
                 SET NOTICE LOGIN DISPLAY

     FILE 'REGISTRY' ENTERED AT 10:19:24 ON 24 SEP 2001
                 SET TERMSET E#
                 DEL SEL Y
                 SEL L14 1 RN
L15            1 S E1/RN
                 SET TERMSET LOGIN

     FILE 'MEDLINE' ENTERED AT 10:19:29 ON 24 SEP 2001
L16           67 S L15
L17           39 S L16 AND PY<=1994
                 E RICE WILLIAM G/AU
                 E RICE W G/AU
L18           51 S E3
L19            0 S L18 AND L16
L20            9 S L18 AND L4
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L24 ANSWER 2 OF 4 MEDLINE
ACCESSION NUMBER: 91288502 MEDLINE
DOCUMENT NUMBER: 91288502 PubMed ID: 2062837
TITLE: Copper inhibits the **protease** from human immunodeficiency virus 1 by both cysteine-dependent and cysteine-independent mechanisms.
AUTHOR: **Karlstrom A R**; Levine R L
CORPORATE SOURCE: Laboratory of Biochemistry, National Heart, Lung, and Blood
Institute, National Institutes of Health, Bethesda, MD 20892.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1991 Jul 1) 88 (13) 5552-6.
Journal code: PV3; 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199108
ENTRY DATE: Entered STN: 19910825
Last Updated on STN: 20000303
Entered Medline: 19910802

AB The **protease** of the human immunodeficiency virus is essential for replication of the virus, and the enzyme is therefore an attractive target for antiviral action. We have found that the viral **protease** is inhibited by approximately stoichiometric concentrations of copper or mercury ions. Inactivation by Cu2+ was rapid and not reversed by subsequent exposure to EDTA or dithiothreitol. Direct inhibition by Cu2+ required the presence of cysteine residue(s) in the **protease**. Thus, a synthetic **protease** lacking cysteine residues was not inhibited by exposure to copper. However, addition of dithiothreitol as an exogenous thiol rendered even the synthetic **protease** susceptible to inactivation by copper. Oxygen was not required for inactivation of either the wild-type or the synthetic **protease**. These results provide the basis for the design of novel types of **protease** inhibitors.

ACCESSION NUMBER: 1999030124 MEDLINE
DOCUMENT NUMBER: 99030124 PubMed ID: 9814959
TITLE: Chemical inactivation of retroviral infectivity by targeting nucleocapsid protein zinc fingers: a candidate SIV vaccine.
AUTHOR: Arthur L O; Bess J W Jr; Chertova E N; Rossio J L; Esser M T; Benveniste R E; Henderson L E; Lifson J D
CORPORATE SOURCE: AIDS Vaccine Program, SAIC/Frederick, National Cancer Institute-Frederick Cancer Research and Development Center, Maryland 21702-1201, USA.
CONTRACT NUMBER: NO1-CO-56000 (NCI)
SOURCE: AIDS RESEARCH AND HUMAN RETROVIRUSES, (1998 Oct) 14 Suppl 3 S311-9.
Journal code: ART; 8709376. ISSN: 0889-2229.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199901
ENTRY DATE: Entered STN: 19990128
Last Updated on STN: 19990128
Entered Medline: 19990111
AB Although most viral vaccines used in humans have been composed of live attenuated viruses or whole killed viral particles, the latter approach has received little attention in research on experimental primate immunodeficiency virus vaccines. Inactivation procedures involving heat or formalin appear to adversely affect the viral envelope proteins. Recently we have inactivated human immunodeficiency virus type 1 (**HIV**-1) with the compound 2,2'-dithiodipyridine (**Aldrithiol**-2, Aldrich, Milwaukee, WI), which inactivates infectivity of retroviruses by covalently modifying the nucleocapsid zinc finger motifs. **HIV**-1 inactivated with **Aldrithiol**-2 retained the conformational and functional integrity of the viral and virion-associated cellular proteins on the viral membrane. We have extended our studies of zinc finger targeted inactivation to simian immunodeficiency virus (SIV) and evaluated the feasibility of applying the procedures to large scale (>30 l) production and purification of the primate immunodeficiency viruses. There was no detectable residual infectivity of SIV after treatment with 1 mM **Aldrithiol**-2 (>5 logs inactivation). Treatment with **Aldrithiol**-2 resulted in extensive reaction with the nucleocapsid protein of treated virus, as shown by immunoblot and high-performance liquid chromatography (HPLC) analysis. As expected, the virion gp120SU appeared to be completely unreactive with **Aldrithiol**-2. Sucrose gradient purification and concentration procedures resulted in little loss of viral infectivity or virion-associated gp120SU. When tested in a gp120-CD4 dependent cell binding assay, the inactivated virus bound to cells comparably to the untreated virus. Analysis of gp120-CD4 mediated postbinding fusion events showed that the inactivated virus could induce CD4-dependent fusion with efficiencies similar to the untreated virus. Inactivation and processing of primate immunodeficiency viruses by methods described here results in highly concentrated virus preparations that retain their envelope proteins in a native configuration. These inactivated virus preparations should be useful in whole killed-particle

vaccine experiments as well as laboratory reagents to prepare antisera, including monoclonal antibodies, and to study noninfective virion-cell interactions.